Arterial injuries in the thoracic outlet syndrome

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Purpose: **This article reviews experience with arterial injury caused by thoracic outlet syndrome. Special emphasis is placed on the influence of athletic or work activities on the axillary-subclavian artery system and the mechanism by which the humeral head compresses the axillary artery and the circumflex humeral arterial branches.**

Methods: **Retrospective review identified 34 patients (age range 13 to 67 years) treated for upper extremity symptoms or ischemic complications of thoracic outlet syndrome from 1983 to 1993. Evaluation included assessment of occupational and recreational activities plus duplex ultrasonography and contrast arteriography with positional maneuvers.**

Results: **Twenty-two patients (27 arms) had subclavian artery injury, which was most commonly caused by compression by a bony abnormality (cervical rib, 16; anomalous first rib, two; cervical rib and anomalous first rib, two). Fourteen of the 27 arms had distal embolization. All 27 had surgical decompression of the subclavian artery; 15 required concomitant arterial reconstruction. Twelve additional patients (nine athletes) had axillary artery involvement, all from arterial compression by the head of the humerus during abduction maneuvers; all had concomitant compression of the posterior circumflex humeral artery. Axillary arterial injury included thrombosis (one), aneurysm (two), and symptomatic extrinsic compression only (nine). Five patients with axillary artery involvement were treated without a surgical procedure; of the remainder, three underwent decompression procedures only, and four had direct arterial repair. In both groups all subclavian and axillary artery reconstructions were patent at last follow-up examination (mean 31 months).**

Conclusion: **Most patients with thoracic outlet syndrome who have arterial involvement have a bony anomaly causing subclavian artery compression. This study demonstrates that humeral head compression of the axillary artery and its circumflex branches is a surprisingly common pathologic mechanism. Awareness of this condition affords a better therapeutic approach to arterial injuries caused by thoracic outlet syndrome. (J VASC SURG 1995;21:57-70.)**

Arterial complications of thoracic outlet syndrome are uncommon, but when they are present they can result in debilitating, limb-threatening complications, if they are not diagnosed and treated in a timely fashion. Arterial disorders of the shoulder girdle comprise a diverse family of related problems afflicting both the subclavian and axillary arteries; most such problems result from a bony anomaly or exaggerated development of the shoulder girdle musculature. This comprehensive article addresses both subclavian and axillary artery disease, even though the axillary artery is not considered in most discussions of the thoracic outlet. Exclusion of the axillary artery based on the exacting anatomic definition of the thoracic outlet would unnecessarily fragment the diagnosis and treatment of these related disorders.

Injury of the subclavian artery caused by compression by a cervical rib, an anomalous first rib, or the anterior scalene muscle is now well recognized.[1,2] Less well understood, though, are the mechanisms by which damage to the axillary artery occurs. Injury of the axillary artery by the pectoralis minor tendon or adjacent nerves has been reported.[3-5] Arterial compression of the third portion of the axillary artery by the head of the humerus was first reported by Lord and Rosati,[6] but has received little attention until the recent study by Rohrer et al.[7] In their study they

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Table I. Subclavian artery involvement—patient occupation

Homemaker	10
Athletes	
Tennis	1
Kayak	1
Karate	1
Musicians	
Cellist	1
Flutist	1
Policewoman*	1
Carpenter	1
Unspecified†	5

*Traumatic shoulder injury.
†One had traumatic shoulder injury.

Table II. Subclavian involvement: arterial disease (22 patients; 27 arms)

	With cervical rib	*No cervical rib*	*Total*
Positional compression only	6	3	9
Aneurysm only, no embolization	3	2	5
Aneurysm and embolization	7	1	8
Thrombosis of aneurysm and embolization	4	0	4
Embolization, no aneurysm	0	1	1

found that compression of the axillary artery is not uncommon in baseball pitchers. As a result of this repetitive compression, arterial thrombosis of the axillary artery has been occasionally noted.[8,9] In addition the anterior and posterior circumflex humeral arteries may also be stretched and compressed by the musculature of the shoulder girdle. Repetitive trauma to these branch arteries may lead to thrombosis or formation of an aneurysm. Subsequent distal embolization may then result in severe hand ischemia. Extensive distal embolization from aneurysms of the circumflex humeral artery has recently been reported in fistball and volleyball players.[9,10]

This article reviews our experience with arterial injury caused by thoracic outlet syndrome compression. Special emphasis is placed on the influence of athletic or occupational activities on the subclavian-axillary arterial system; also, the mechanism of humeral head compression of the axillary artery and circumflex humeral branches is explored.

MATERIAL AND METHODS

Thirty-four patients were evaluated for arterial problems arising from thoracic outlet syndrome at the Northwestern University McGaw Medical Center Hospitals during the period between October 1983 and November 1993. Nineteen men and 15 women with a mean age of 32 years (range 13 to 67 years) were studied. Diagnostic evaluation included a thorough history, physical examination, and assessment of occupational and recreational activities. In addition to palpation of pulses and auscultation for bruits at neutral, adduction, abduction-external rotation, and specific provocative positions, all patients were evaluated with segmental pressure measurements of the brachial, ulnar, and radial arteries with the Doppler technique described previously.[11] Arterial photoplethysmographic (PPG) studies of the digits, duplex ultrasonography scanning of the subclavian and axillary arteries, and contrast arteriography were also performed with the patient in the neutral position and with positioning maneuvers. Chest and cervical spine radiographs were obtained to define the presence of a bony abnormality such as a cervical rib, anomalous first rib, or healed clavicular fracture. Treatment was based on the mechanism of arterial damage, the degree of ischemia, and the nature of any arterial disease. Follow-up information was obtained with outpatient office visits or telephone interviews. Repeat Doppler segmental limb pressures and duplex ultrasonography examinations were obtained when appropriate. Arterial involvement was readily separable into primary subclavian artery lesions or primary axillary artery disease. The anatomic relationships of the subclavian, axillary, and circumflex humeral branches were studied with the use of fresh-frozen cadaver shoulder specimens; interactive relationships between the arteries and adjacent musculoskeletal structures were examined in both neutral and hyperextension/hyperabduction positions.

Group I: subclavian artery

Subclavian artery injury occurred in 22 patients and involved 27 arms. Nine male patients and 13 female patients ranging in age from 13 to 67 years (mean 35 years, median 36 years) were in this group. The right side was involved in 12 patients, the left side was involved in five patients, and five patients had bilateral involvement. A cervical rib was documented in 16 (73%) of the 22 patients; five of these 16 patients had bilateral cervical ribs. Occupations are outlined in Table I. Of 22 patients nine had a normal result of an arterial examination at neutral position but demonstrated compression with positioning maneuvers. Whereas obliteration of the forearm pulses is known to occur in some healthy individuals, these patients with symptoms were found to have abnormal results of digital arterial PPG studies with the provocative maneuvers. Subsequent

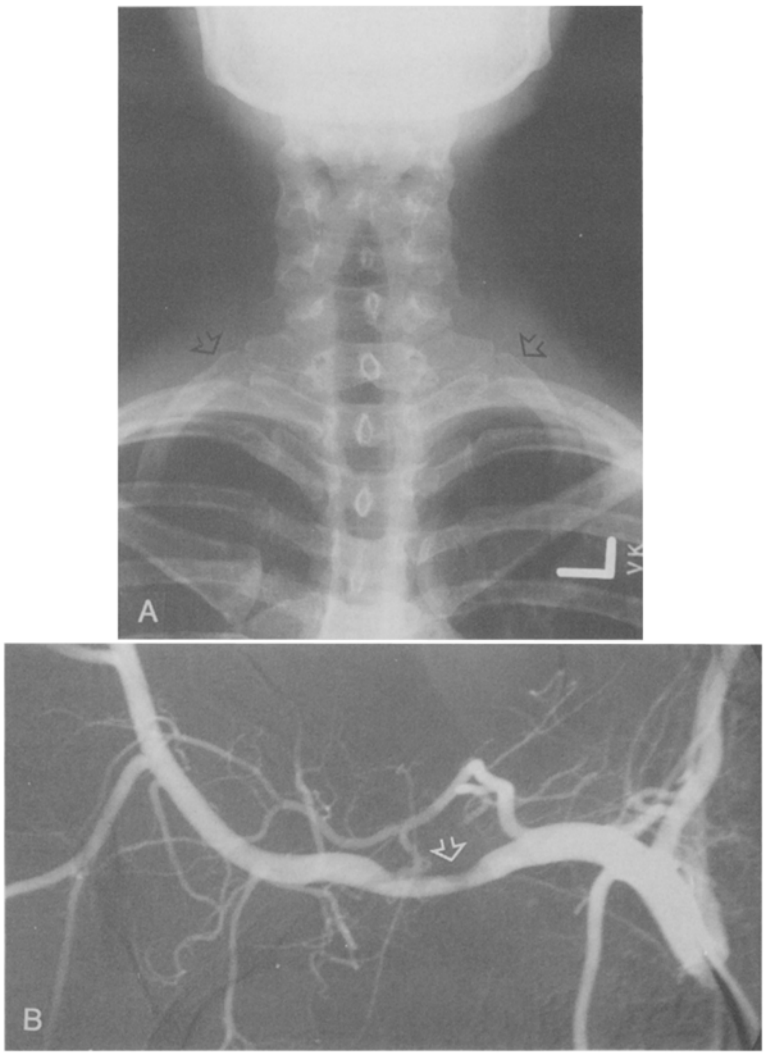

Fig. 1. A, PA radiograph demonstrates bilateral anomalous first ribs *(arrow)*. **B,** Angiogram of site of impingement *(arrow)* on superior aspect of right subclavian artery by anomalous, incomplete first rib with abnormal attachment to underlying second rib during hyperabduction maneuver. Note mild poststenotic dilation.

duplex ultrasonography visually documented actual arterial compression during hyperabduction/hyperextension positioning.

Poststenotic dilatation without embolization occurred in seven upper extremities. Arteriographic evidence of distal embolization to radial, ulnar, palmar, and digital arteries was found in six limbs with subclavian artery aneurysms and in one arm with irregularity of the arterial wall. As a result of delayed diagnosis four patients had already sustained thrombosis of the aneurysm with extensive embolization. Table II depicts the mode of presentation and arterial disease in these patients.

Subclavian artery involvement is most commonly caused by the presence of a cervical rib. An isolated cervical rib was present in 13 limbs of the 22 patients. Two limbs had a combination of cervical rib and anomalous first rib compression, three limbs had a combination of cervical rib and anterior scalene muscle compression, and two limbs had compression by a cervical rib and an anatomically normal first rib. Of those seven limbs with symptoms and no cervical rib, two sustained arterial compression from an anomalous first rib (Fig. 1). Anomalous, or incomplete, first ribs usually have abnormal attachments to the underlying second rib. The remaining five limbs

Table III. Subclavian artery involvement: mechanism of arterial compression (22 patients; 27 arms)

	With cervical rib	*No cervical rib*	*Total*
Cervical rib	13	0	13
Cervical rib and anterior scalene muscle	3	0	3
Cervical rib and first rib	2	0	2
Anomalous first rib	2	2	4
Scalene muscles	0	4	4
Aneurysm of suprascapular artery	0	1	1

Table IV. Axillary artery involvement—patient occupation/activity

Athletes	
Major league baseball pitcher	6
College softball pitcher	1
Competetive high-school swimmer*	1
Amatuer weight-lifter	1
Mechanic	2
Construction worker†	1

*History of clavical fracture.
†Traumatic shoulder injury.

with symptoms had subclavian artery involvement caused by the scalene muscles, the combination of the scalene muscle and anomalous fibrocartilaginous bands, or aneurysmal involvement of the suprascapular artery (Table III). Operative decompression of the artery was performed to address the upper extremity symptoms and to prevent arterial damage.

The operative approach for the treatment of subclavian artery compressive symptoms or actual arterial damage with complications requires removal of the offending compressive element, whether this element is soft tissue or a bony structure. For those patients with severe compressive symptoms and no arterial damage, mere decompression of the artery with close operative inspection is appropriate. Similarly mild poststenotic ectasia does not require repair of the artery; decompression of the site of impingement and close noninvasive follow-up examinations have yielded good results to date. Intraoperative B-mode ultrasonography may be helpful in the assessment of intimal involvement. However, actual aneurysmal degeneration of the artery or intimal damage (with or without embolic sequelae) requires resection of the damaged artery by arterial reconstruction with the use of a saphenous vein graft. We preferred to use a two-incision technique (supraclavicular and infraclavicular), and this technique has been described in detail previously.[12] In essence the supraclavicular approach allows the division of the anterior scalene muscle, facilitating the exposure of the proximal subclavian artery. An infraclavicular incision is used for exposure of the first and second portion of the axillary artery. The pectoralis minor tendon may be divided to facilitate exposure of the distal axillary artery. When a cervical rib is present excision of the cervical rib and any abnormal attachments is performed. The transaxillary approach for resection of the first rib was used to decompress the artery when arterial involvement was caused by true thoracic outlet compression, and no direct exploration or reconstruction of the subclavian or axillary artery was required.

All 22 patients (27 arteries) underwent a surgical procedure for decompression or repair of a subclavian artery. Two patients required simultaneous thromboembolectomy of the distal circulation of the forearm. Three additional patients required a distal bypass (subclavian-brachial, one; axillary-brachial, two) to treat chronic forearm ischemia. The greater saphenous vein was the conduit of choice; one female patient required polytetrafluoroethylene interposition graft replacement of the subclavian artery because of a small saphenous vein of inadequate caliber.

Group II: axillary artery

Twelve patients (14 arms) presented with primary involvement of the axillary artery. Ten men and two women ranging in age from 15 to 37 years (mean 26 years, median 27 years) were in this group. No osseous abnormalities were found. Of the 12 patients nine had unilateral right extremity involvement, whereas three had bilateral involvement. All patients were either manual laborers or advanced athletes. Of the nine athletes six were major league baseball pitchers, and one was a varsity intercollegiate softball pitcher (Table IV). Six of the seven pitchers had symptoms of pain, weakness, or tingling of the fingers that affected their pitching control or endurance. Two patients had undergone thrombolytic therapy for axillary artery thrombosis before presentation. Only one pitcher had no discrete symptoms; his axillary artery compression was detected from a bruit found on physical examination. The remaining two athletes who presented with an early onset of arm fatigue both had significant development of upper body musculature, one from competitive high-school varsity swimming and the other from weight lifting. The three nonathletes included two mechanics and a construction worker who had sustained a work-

related shoulder injury with subsequent neurologic symptoms. One mechanic had bilateral pulsatile axillary masses, and the other presented with bilateral arm fatigue after an automobile accident (Table V).

Initial noninvasive assessment included segmental Doppler arterial blood pressures at rest. Subsequent digital PPG studies were obtained during provocative maneuvers (hyperabduction/hyperextension of the upper extremity) to document proximal compression of the arterial system. Abnormal PPG results led to Duplex ultrasonography study of the axillary and subclavian arteries. Proximal compression of the subclavian artery by soft tissues or bony structures is readily appreciated and documented by the experienced ultrasonographer. Failure to identify compromise of the subclavian artery leads to study of the axillary artery at neutral and provocative positions. The duplex ultrasonography image can define compression of the third portion of the axillary artery by the head of the humerus during provocative maneuvers, because direct compression of the artery by the bright echogenic cortex of the humeral head is readily evident on appropriate views. Subsequent arteriography confirmed compression of the axillary artery during abduction-external rotation of the upper extremity. This compression was consistently localized to the third portion of the axillary artery and appeared to be due to compression of the artery by the head of the humerus during 90 degrees of abduction and external rotation (Fig. 2). All of these patients also had angiographic occlusion of the apparently normal posterior circumflex humeral artery during the provocative positioning maneuver (Fig. 3).

The surgical exposure of the third portion of the axillary artery is performed through a transaxillary approach. The patient is placed in the supine position with the arm abducted and prepped in the operative field. Having the arm prepped and draped freely into the operative field allows full range of motion during the operation to assess the adequacy of arterial decompression in all positions. A transverse incision is made just below the axillary hairline (Fig. 4). The incision extends from the posterior border of the pectoralis major to the latissimus dorsi posteriorly. The subcutaneous tissue is mobilized into the axilla. During this portion of the dissection the intercostal brachial nerve is identified and preserved, if possible. The patient is forewarned of potential numbness in the distribution of this nerve should it be necessary to resect it. After the neurovascular bundle is exposed, the axillary artery is traced proximally. The pectoralis minor and major muscles are retracted, providing exposure to the junction of the second and third portion of the axillary artery. The posterior circumflex humeral artery and the subscapular artery are important landmarks to identify the area of arterial compression (Fig. 5). At this level the brachial plexus encircles the axillary artery, and the most inferior branch is the ulnar nerve. The axillary nerve and the posterior circumflex humeral artery course posteriorly through the quadrilateral space. The quadrilateral space (an area of muscular entrapment) is formed by the teres major, the biceps brachii, the humerus, and the subscapular muscle. In the professional athletes the subscapular and circumflex humeral arteries were hypertrophied, and in one patient they had become aneurysmal.

Careful delineation of the arterial anatomy is essential at this point, because the posterior circumflex humeral and anterior circumflex humeral arteries may arise from a common trunk.[13] Ligation of the anterior circumflex humeral artery is best avoided, because this artery provides the main source of blood supply to the head of the humerus.[14] No attempt is made to release the quadrilateral space, because this would require division of the tendinous head of the teres major and triceps brachii muscles. This axillary approach affords reasonable visualization of both the second and third portions of the axillary artery and avoids muscle transection, which could be debilitating. More proximal exposure is difficult because of the overlying pectoralis musculature.

Depending on the arterial disease, the axillary artery is either repaired directly or patched, or the branch arteries are ligated and divided. Virtually all axillary artery problems appeared to be due to compression of the third portion of the artery by the head of the humerus. Because the offending bony structure, the head of the humerus, obviously cannot be removed, modification of the artery is required. For those patients who have compressive symptoms only and no arterial damage, vein patch angioplasty of the axillary artery at the site of compression enlarges the effective diameter of the artery, allowing continued blood flow past the affected site during provocative maneuvers. Once actual arterial wall damage has occurred, that portion of the axillary artery must be replaced; an interposition graft of autogenous saphenous vein removes the arterial disease and allows anatomic routing of the artery around the site of humeral head compression. Preservation of the circumflex humeral arterial branches should be attempted, if possible, by reimplantation. Aneurysmal degeneration of the circumflex humeral arteries presents a challenge; arterial reconstruction of these important branches is usually not possible,

Table V. Axillary artery involvement (12 patients; 14 arms)

Sport/occupation	*Sex*	*Age*	*Signs and Symptoms*	*Angiographic findings*
Baseball pitcher, major league	M	25	Pain in shoulder while pitching	Humeral head compression
Swimmer, high-school	F	15	Pain in shoulder and forearm fatigue	Humeral head compression, circumflex artery occlusion
Mechanic A	M	37	Fatigue of both arms after car accident	Humeral head compression, circumflex artery occlusion
Weight-lifter, amateur	M	29	Fatigue right arm with exertion	Humeral head compression
Baseball pitcher, major league	M	26	Distal limb ischemia from embolization	Aneurysm of posterior circumflex humeral artery; embolic occlusion of brachial, radial, and ulnar arteries
Construction worker A	M	27	Neurologic symptoms after work injury, right arm	Compression of subclavian artery by scalene muscle; compression of axillary by humeral head
Construction worker A	M	27	Neurologic symptoms after work injury, left arm	Compression of subclavian artery by humeral head
Baseball pitcher, major league	M	23	Pain over deltoid; numbness in fingers	Humeral head compression; circumflex artery occlusion
Baseball pitcher, major league	M	28	Weakness, numbness, and pain of right hand	Humeral head compression
Baseball pitcher, major league	M	31	Arterial bruit; compression by duplex scanning	Humeral head compression
Baseball pitcher, major league	M	27	Severe hand ischemia; previous lytic therapy and radial artery embolectomy	Humeral head compression
Mechanic B	M	27	Pulsatile mass, left axillary artery	Mild ectasia distal to anterior scalene muscle and first rib; posterior humeral circumflex artery occlusion
Mechanic B	M	27	Pulsatile mass, right axillary artery	Axillary aneurysm; posterior humeral circumflex artery occlusion
Softball pitcher, college	F	18	Cold hand caused by axillary thrombosis; successful lytic therapy	Humeral head compression

M, male; *F*, female.
*Still has axillary artery compression by duplex scanning.

because it requires exclusion of the aneurysmal segment and carries the theoretic risk of devascularization of the humeral head.

RESULTS

Within the entire cohort 29 of the 34 patients underwent operation. No deaths or amputations occurred during operation. Follow-up examination has ranged from 1 month to 9 years, 5 months (mean 31 months). Periodic surveillance with segmental Doppler limb pressures and duplex ultrasonography scanning not only confirms adequate functional outcome but also allows for assessment of any potential recurrent compressive complications.

Group I: subclavian artery

All patients in this group underwent operation for arterial decompression. Removal of the compressive structure, whether it was a rigid bony component or a soft-tissue element, typically resulted in relief of symptoms and improved noninvasive studies. Normal or mildly ectatic, nonaneurysmal arteries were simply freed from compressive structures to prevent further aneurysmal degeneration. Repair of any damaged arterial segment (aneurysm or intimal damage) ensured prevention of any further upper extremity ischemic complications. Fifteen subclavian artery reconstructions were performed. Three required concomitant distal bypass, and two required adjunctive brachial thromboembolectomy and patch angioplasty. One patient required ligation and excision of a suprascapular artery aneurysm. The remaining six patients required no vascular reconstruction to complement the removal of the compressive structures. Two of the vascular reconstructions have required reoperation. One patient experienced thrombosis of a polytetrafluoroethylene interposition graft that had been placed to repair a subclavian artery

Treatment	*Outcome*	*Follow-up (mo)*
Change in pitching motion	Pitching successfully	22
Exploration and patch angioplasty	Swimming competitively	29
Observation	Awaiting surgical procedure	14
Artery exploration; division of pectoralis minor	Somewhat better*	6
Patch angioplasty; excision of branch aneurysm; brachial-interosseous bypass	Pitching well	35
Anterior scalenectomy; partial middle scalenectomy*	Required patch angioplasty 4 months later	14
Anterior scalenectomy	Doing well; back at work	4
Change in pitching motion	Pitching; still has symptoms*	12
First rib resection; exploration of artery	Better, but not pitching	5
Observation	Still pitching	33
Patch angioplasty, third portion axillary artery; exclusion of posterior circumflex branch	Not pitching; no ischemic symptoms	32
Anterior scalenectomy	Back at work	9
Resection of aneurysm and bypass graft	Back at work	8
Modification of pitching motion	No better; surgical procedure planned 6/94	6

aneurysm caused by a cervical rib (which had been excised). Thrombosis occurred on postoperative day 82 and was successfully treated by graft thrombectomy; the graft was patent at 78 months. The second patient had aneurysmal degeneration of a reversed saphenous vein interposition graft that had been used to replace a subclavian artery aneurysm 59 months previously. The aneurysmal graft was replaced by a new segment of reversed saphenous vein, which remains patent without complication at 55 months after operation. The use of greater saphenous vein for patch angioplasty or bypass conduit has resulted in 100% patency, with no evidence of recurrent embolization at mean follow-up time of 38 months (range 1 to 113 months).

Group II: axillary artery

In contrast to the patient with subclavian artery injury, the approach to the patient with axillary artery involvement is different by necessity. Because the mechanism of arterial compression is most often the head of the humerus, resection of the bony structure cannot be performed. Four pitchers were treated by modification of their pitching mechanics; two were able to continue pitching successfully, but one has retired from baseball, and the other is presently awaiting surgical correction for persistent symptoms. Of the three pitchers treated surgically one continues to excel in the major leagues, whereas the other two have retired without further symptoms. After surgical procedures the swimmer continues to compete successfully, and the weight lifter's condition is improved. The construction worker and one mechanic underwent surgical correction, and both are back at work; the second mechanic is awaiting surgical therapy. Five patients required reconstruction of the axillary artery. Four arteries were treated by vein patch angioplasty, one of which had a simultaneous brachial-interosseous bypass for embolic occlusion of the brachial, radial, and ulnar arteries. The remaining reconstruction was a vein interposition graft for intimal disruption of the axillary artery (Fig. 6). All five vascular reconstructions were patent at last evaluation (mean follow-up time 18 months; range 5 to 35 months).

One of the five axillary artery reconstructions required reoperation. The perioperative use of systemic heparin resulted in an arm hematoma that led to compression and thrombosis of a brachial-interosseous artery bypass (reversed saphenous vein) that had been performed as an adjunct to the repair of an axillary artery branch aneurysm. Evacuation of the hematoma and thrombectomy of the vein graft successfully restored normal blood flow, and the bypass was patent at 3 months.

Only one patient has required reoperation for inadequate surgical decompression of the thoracic outlet or shoulder girdle. A construction worker with traumatic thoracic outlet syndrome underwent anterior scalenectomy and partial middle scalenectomy to treat compression of the subclavian and axillary arteries by the scalene muscles and the humeral head. Two months later repeat duplex ultrasonography scan confirmed continued compression of the axillary artery by the head of the humerus. Exploration of the first, second, and third portions of the axillary artery combined with vein patch angioplasty of the compressed arterial segment has led to resolution of symptoms 8 months later.

DISCUSSION

Thoracic outlet syndrome is a family of disorders that affect the subclavian-axillary neurovascular bundle as it exits the thoracic cage. Many symptoms

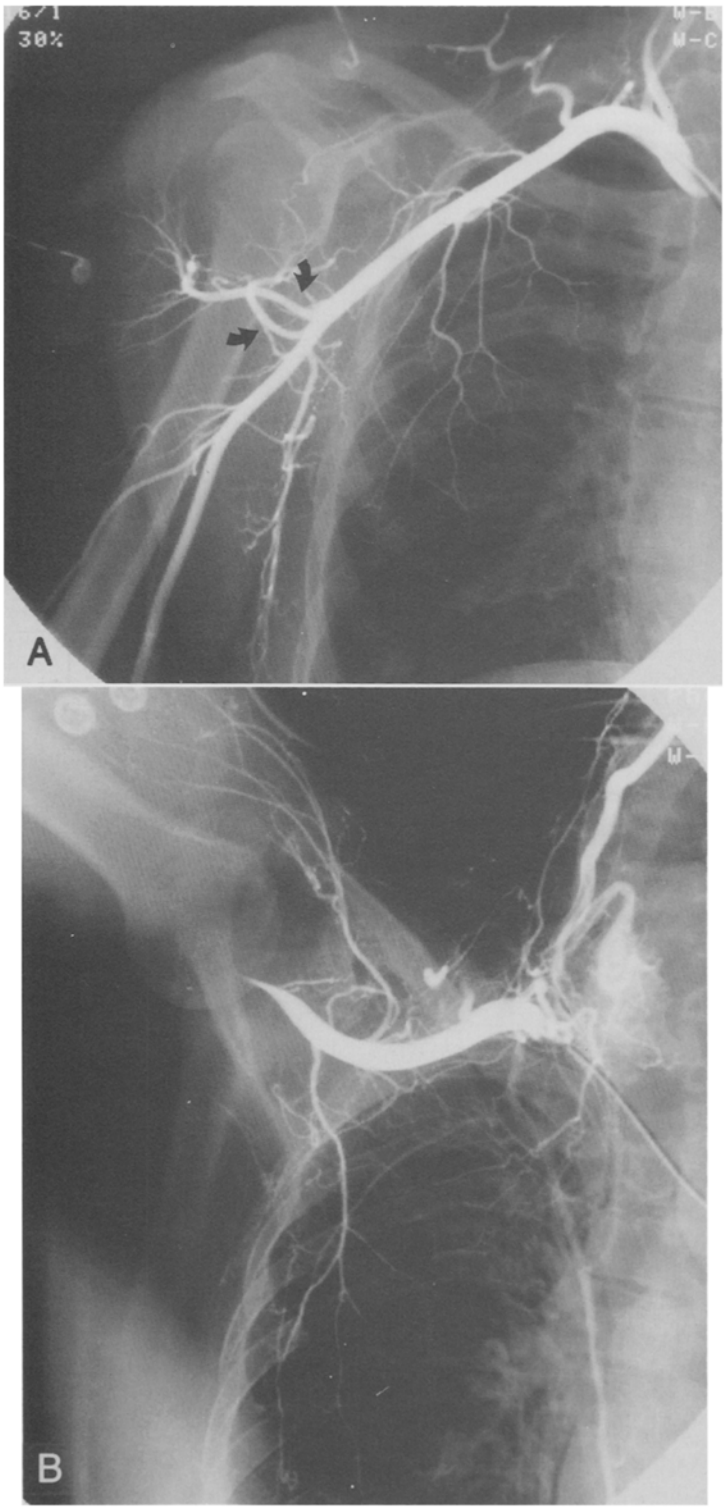

Fig. 2. A, Right subclavian-axillary angiogram of weight-lifter in neutral position. Note generous size of circumflex humeral arteries *(arrows)* and arterial loop they form around surgical neck of humerus. **B,** Angiogram of same patient during hyperabduction-external rotation maneuver. Note beaklike occlusion of axillary artery at level of humeral head. Circumflex humeral arteries are also occluded.

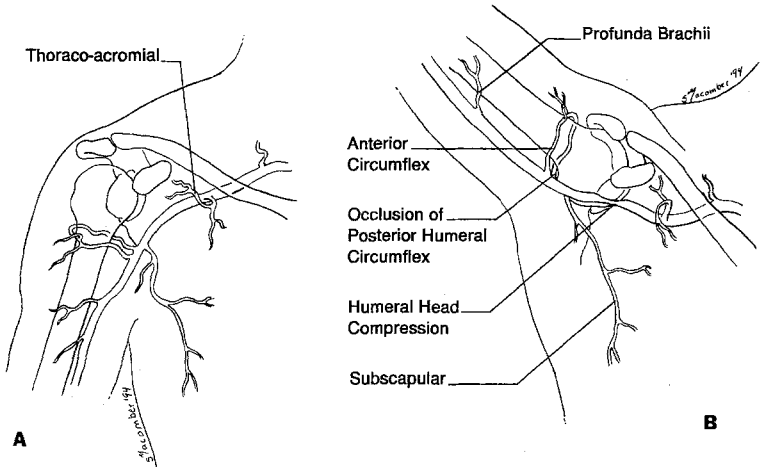

Fig. 3. A, Diagrammatic representation of axillary artery, its major branches, and adjacent bony structures in neutral position. **B,** Depiction of compression of axillary artery and posterior circumflex humeral artery by humeral head as arm is abducted and externally rotated. Branch arteries tether axillary artery in fixed position, leading to compression by downward displacement of humeral head. Note arterial loop that is formed by anterior and posterior circumflex humeral arteries.

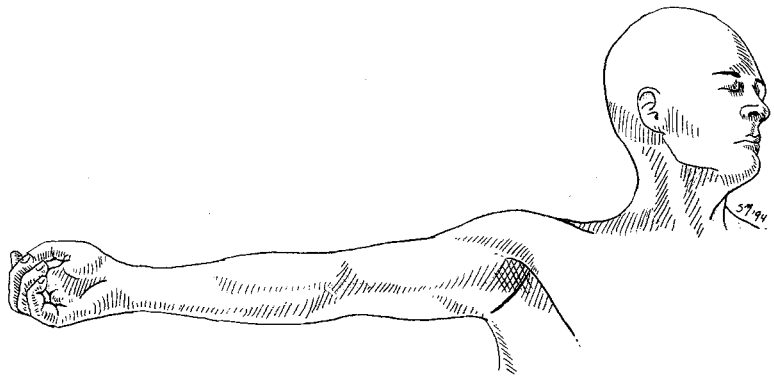

Fig. 4. Operative positioning of patient: supine position with upper extremity abducted and prepared into operative field. Note site of transverse incision just below axillary hairline.

are due to compression of the neurologic and venous structures, and arterial complications at this site are not common. Since Mayo[15] and Murphy[16] made the first reports of the association between a cervical rib and a subclavian artery aneurysm, the awareness and diagnosis of arterial injuries of the thoracic outlet at any given time have paralleled the state of knowledge of the various causative mechanisms.[2] Subsequently described mechanisms all result in compression of the neurovascular bundle by components of the thoracic outlet and shoulder girdle. Anatomic mechanisms of compression have been found to result from the scalene muscles, the clavicle and first rib, fibrocartilaginous bands, anomaly of the first rib, and clavicular fractures. These diverse components all result in a similar pathologic condition: compression of the axillo-subclavian neurovascular bundle between the rigid structures. Successful therapy requires precise localization and definition of the arterial disease and identification of the causative compressive elements.

The diagnosis and treatment of these disorders require a high level of suspicion. In patients thought to have thoracic outlet compression, the use of duplex scan or arteriography including positional views helps to establish the diagnosis. Four patients in this series had a delay in diagnosis, which resulted in arterial thrombosis and extensive distal embolization. The delay of diagnosis is often due to incomplete

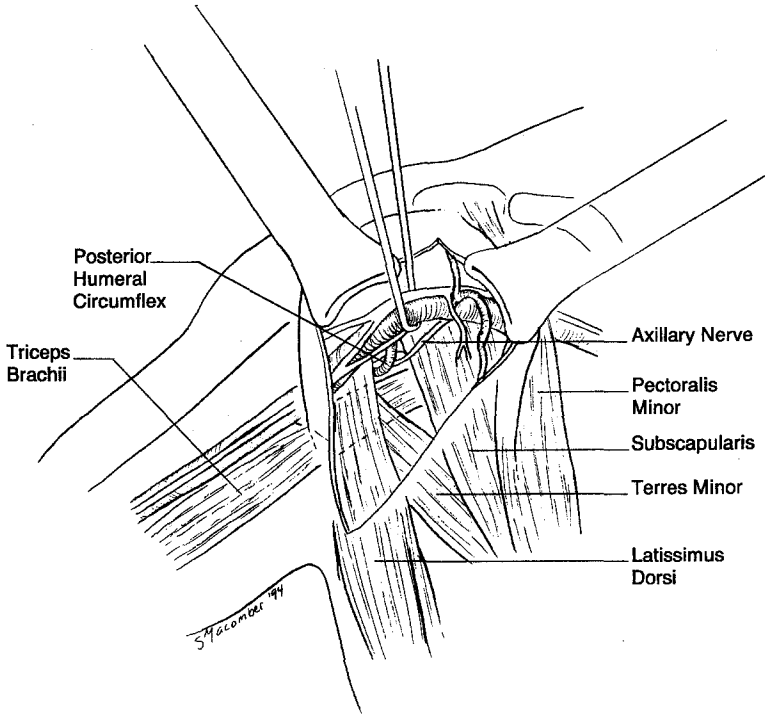

Fig. 5. Diagrammatic representation of third part of axillary artery and its branches as exposed through described axillary incision approach.

arteriographic examination of the proximal subclavian artery and its relation to abduction-external rotation or hyperabduction maneuvers. In the arteriographic evaluation of these patients it is also important to identify disease of the major branches. In a previous report distal embolization occurred from a supraclavicular artery.[8] Here we report the disease of the posterior circumflex humeral artery with aneurysm formation and distal embolization.

Arterial injuries of the subclavian artery in the thoracic outlet are generally produced by osseous abnormalities. As in this series the literature reports that most patients (70% to 100%) presenting with subclavian artery injuries within the thoracic outlet have cervical ribs.[17-22] In this series 73% of the patients were found to have a cervical rib. In addition anomalies of the first rib (as in four of our patients) may produce arterial injury but are uncommon in most series.[23,24] With cervical ribs the subclavian artery passes over the bony abnormality, compresses the inferior aspect of the artery, and produces an intimal lesion with or without a poststenotic dilatation. The presence of an anomalous first rib can cause similar arterial changes but impinges on the superior aspect of the artery (Fig. 1). Unfortunately these patients often present with distal embolism as the initial symptom. Distal embolism is the most debilitating aspect of this disease and is frequently difficult to treat. Uncommonly a normal anatomic muscular structure may produce significant arterial injury. Hypertrophy of the anterior scalene muscle in athletes has been associated with arterial compromise of the subclavian artery and occlusion of the supraclavicular artery with distal embolization.[8] Four patients in this series presented with no bony abnormalities despite evidence of distal embolization or arterial compression. Recently Cormier et al.[1] reported the long-term results of 55 procedures for arterial injuries of the subclavian artery associated with the thoracic outlet. Distal embolism had occurred in 35 (64%) patients. Proximal emboli were removed with embolectomy catheters, whereas distal emboli were not treated. In 39 of the patients in whom long-term follow-up was available, no amputations occurred, and 90% of these patients had no symptoms. Four patients had symptoms including forearm pain with exertion akin to claudication. Kieffer and Ruotolo[17] also reported excellent results in 97% of patients, noting only a single digital amputation (mean follow-up time 43.7 months) and a

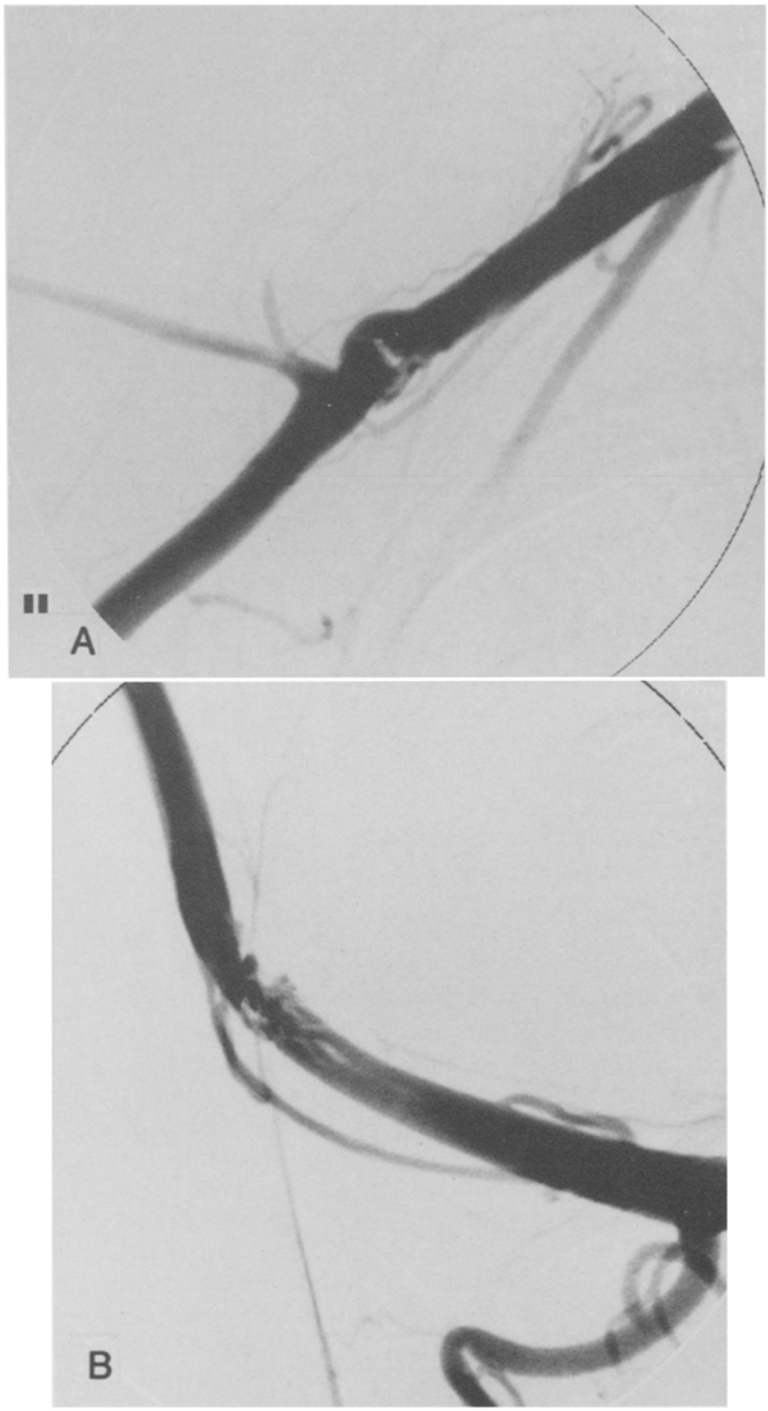

Fig. 6. A, Axillary arteriogram in neutral position depicts intimal fracture of third portion of axillary artery. **B,** Same patient during abduction with accentuation of intraluminal irregularity that had resulted in distal embolization. Note that posterior humeral circumflex artery has become occluded during maneuver.

graft occlusion in one patient. Those results are similar to those obtained in this series, which involved a similar supraclavicular and infraclavicular approach. However, a different operative approach has been reported by Green et al.[25] With their approach the medial half of the clavicle is resected. This approach allows direct access to the subclavian artery directly beneath the clavicle. The authors report no significant long-term disabilities from this approach.

Compression of the subclavian artery by musculoskeletal structures is a well-documented feature of thoracic outlet syndrome; however, little information exists that relates a mechanism of injury to the axillary artery and its major branches. The first recognition that the head of the humerus could compress the axillary artery was made by Lord and Rosati in 1958.[6] Angiographic documentation of this compressive phenomenon was published in 1978 by Dijkstra and Westra,[26] who demonstrated angiographic occlusion of an apparently normal axillary artery by humeral head compression with a 100-degree abduction maneuver. More recently Rohrer et al.[7] have studied and suggested the role of the humeral head in axillary artery compression and injury in throwing athletes. In our previous article, we also encountered humeral head compression of the axillary artery, particularly the posterior humeral circumflex branch of the artery.[8] In the current analysis all 12 patients with humeral head compression of the axillary artery also had compression of the posterior circumflex humeral artery. This arteriographic pattern is similar to the "quadrilateral space syndrome" in baseball pitchers described in the orthopedic literature.[27,28] The act of pitching results in exaggerated shoulder movements as shown by detailed studies of the biomechanics of pitching.[29] During the "cocking phase" of pitching the shoulder often reaches a position of approximately 90 degrees abduction, 30 degrees horizontal extension, and 90 to 120 degrees external rotation. This extreme humeral rotation causes downward displacement of the humeral head with resultant compression of the axillary artery. These forceful motions are repeated innumerable times on a daily basis by an active baseball pitcher. A possible contributing factor may be the finding that humeral head hypertrophy occurs with vigorous upper limb exercise.[30] An increase in humeral head diameter would probably lead to increased stretch and tension of the axillary artery during extreme pitching motions. With natural hypertrophy of the shoulder girdle musculature in a major league pitcher, the circumflex humeral and subscapular artery branches become greatly enlarged (reaching a diameter of 7 to 8 mm in one of the pitchers operated in this series).

Extension and abduction maneuvers of the upper limb lead to downward displacement of the humeral head. We believe that this displacement creates a traction mechanism that leads to the fixation or tethering of the axillary artery by the circumflex branches as it winds and stretches across the head of the humerus (Fig. 3,*B*). Lord and Rosati[6] believed that full hyperabduction of the arm over the head pulls the axillary artery across the coracoid process and the head of the humerus, "as across a pulley." This stretching effect on the axillary artery leads to localized damage at the site of contact with the humerus head. Because of repetitive shoulder motion the circumflex humeral arteries themselves may also be subjected to chronic irritation leading to occlusion or aneurysm formation. The circumflex humeral arteries arise from the third portion of the axillary artery and wind around the surgical neck of the humerus, supplying the nutrient blood flow to the head of the humerus. The arterial loop that is thus formed by the anterior and posterior circumflex humeral arteries is stretched with external rotation/hyperabduction of the arm (Fig. 3,*B*). This repetitive tension effect (as in a taut, stretched rubber band) may lead to intimal fracture or damage and weakening of the wall of these branch arteries and the subsequent aneurysmal degeneration that has been documented in our athletic patients. Further support of this concept is found in recent reports of aneurysm formation in circumflex humeral artery branches in volleyball and fistball players.[9,10]

Effective treatment of thoracic outlet syndrome arterial injuries requires early recognition of the arterial complication and prompt correction of the compressive mechanism. The treatment of subclavian artery injury is now well established. In this series all patients experienced excellent outcomes after surgical procedures to correct an osseous anomaly and after subclavian artery repair. On the other hand the definitive treatment for humeral head compression of the axillary artery is an evolving process. Certainly when injury of the axillary artery or one of the circumflex humeral branches occurs, a reconstructive surgical procedure is necessary. The third portion of the axillary artery is best approached as described in this article. For athletes with compression alone and no structural injury or embolization, the wisdom of direct surgical intervention remains uncertain. Com-

pression of the axillary artery by the head of the humerus may not be a pathologic symptom but may be a significant warning sign that the axillary artery in that extremity is being abnormally stressed. For the baseball pitcher a modification of pitching mechanics should be tried first; surgical intervention should be reserved for persistent symptoms or ischemic complications. Close periodic surveillance with arterial examination and duplex ultrasonography scanning should be instituted to detect early signs or symptoms of aneurysmal degeneration, intimal damage, embolization, or branch artery involvement in these athletes. Persistence or worsening of symptoms may require operative intervention. In the absence of intimal damage, thromboembolic complications, or aneurysm formation, surgical therapy is aimed at slightly enlarging the effective diameter of the axillary artery at the site of humeral head compression; vein patch angioplasty with the greater saphenous vein yields excellent results. Intimal damage or aneurysmal change of the artery mandates replacement of the injured segment with an interposition graft of reversed saphenous vein. Judicious surgical intervention and close follow-up examination yield gratifying long-term results in this fascinating group of patients.

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DISCUSSION

Dr. Herbert I. Machleder (Los Angeles, Calif.). Dr. Durham and the vascular group at Northwestern University have provided us with a very valuable and practical article dealing with embolizing or thrombotic arterial lesions from repetitive compression at the thoracic outlet. They emphasize a number of observations that are consonant with our own experience.

Without a high index of suspicion these lesions are apt to be overlooked. Even when the diagnosis is suspected, the evaluation is often inadequate. They stress the importance of examining the upper extremity arterial system both clinically and radiologically in the neutral and in the stress positions. In fact 41% of the embolizing subclavian abnormalities were unassociated with evidence of compression in the neutral position.

The authors further observed that arterial injuries can occur as a consequence of musculotendonous compression. For example 27% of subclavian compressive injuries occurred in the absence of a cervical rib.

With regard to screening noninvasive tests, we found that digital photoplethysmography is most useful (particularly in throwing athletes with symptoms), facilitating observation of pulse changes in the full range of stress positions. Could the authors comment on their initial diagnostic protocol?

I have several questions with reference to their treatment strategies. Many of these patients are referred for evaluation and treatment when multiple emboli have obstructed the digital or palmar vessels with consequent ischemic symptoms. In the proximal repair you are essentially arresting further embolization rather than correcting that which has already occurred. How do you deal with symptomatic distal emboli? At the time of proximal repair what is the role of sympathectomy for the painfully ischemic hand or fingers?

In our experience the thrombus often extends distally and proximally, obscuring the exact site of injury. After thrombolytic therapy the artery can appear surprisingly normal, until it is viewed in the stress position. I think it worth emphasizing that the abnormalities causing arterial damage in the first, second, and third portions of the subclavian artery and in the first, second, and third portions of the axillary artery are different and unique. Is this consistent with your observations? How do you view the role of thrombolytic therapy in your management sequence?

You indicated that poststenotic dilatation without embolization occurred in seven patients. Is decompression of the thoracic outlet without direct arterial repair a reasonable approach in these patients? And what is the natural history of the ectasia?

The therapeutic strategy for the third portion of the axillary artery seems to me to be the most difficult, particularly compression or attenuation of the artery over the subluxing head of the humerus. You indicated that four of the five reconstructions in this area were vein patch angioplasties. I wonder whether you could describe the procedure and rationale in a bit more detail.

I have one final question. In our series at UCLA we have been concerned about costoclavicular passage of grafts in these cases of compressive thrombosis. Consequently we remove the first rib, enlarging both the interscalene and costoclavicular space. How do you ensure adequate decompression such that when the patient returns to normal activity, the graft does not share the fate of the injured artery?

Once again I would say that the authors have made a substantial contribution to our understanding of these difficult disorders.

Dr. Joseph R. Durham. The initial noninvasive evaluation includes segmental Doppler studies and digital PPG studies in neutral and stress positions. We use selective preoperative and intraoperative lytic therapy to treat symptomatic distal thromboembolic problems much more now than in the study's early stages. We have not used sympathectomy, relying rather on surgical reconstructions to address ischemic problems of the hand or digits.

Because there are many mechanisms of arterial compression at the thoracic outlet, each approach must be tailored to the unique anatomy of a given patient. This may require first rib or cervical rib resection in addition to arterial reconstruction. Some patients may require decompression of the thoracic outlet without the need for direct arterial repair; these are typically younger patients with arterial ectasia rather than true aneurysmal degeneration. The natural history of such ectasia is not known, so close follow-up is essential.

Vein patch angioplasty to treat compression of the third portion of the axillary artery is used to enlarge the effective arterial diameter, because removal of the compressing humeral head is not practical. The location of the circumflex humeral arteries helps to pinpoint the exact site of arterial involvement. Excellent results have been obtained with the use of the greater saphenous vein for patch angioplasty. Intraoperative full-range manipulation of the shoulder allows assurance that arterial decompression is complete.